## Refine Search

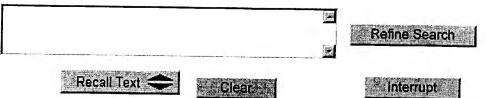
### Search Results -

	Documents
(19 NOT 20).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	63
(L19 NOT L20 ).PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD.	63

Database:

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US Patents Full-Text Database
US OCR Full-Text Database
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Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search:



### Search History

DATE: Tuesday, October 26, 2004 Printable Copy Create Case

Set Name side by side DB=P OP=AN	PGPB, USPT, USOC, EPAB, JPAB, DWPI, TDBD; THES=ASSIGNEE: PLUR=3	Hit Count	Set Name result set
<u>L21</u>	L19 not L20	63	L21
<u>L20</u>	L19 and L17	8	L20
<u>L19</u>	L2 same (proliferation)	71	<u>L</u> 19
<u>L18</u>	L17 not L11	265	L18
<u>L17</u>	L16 and L14	391	<u>L</u> 17
<u>L16</u>	(inhibit or reduce) same (proliferation)	25725	L16
<u>L15</u>	L14 and L6	13	L15
<u>L14</u>	L13 and L3	555	<u>L14</u>
<u>L13</u>	L12 and L2	1348	<u>L</u> 13
<u>L12</u>	(screening or testing or identifying) same (drug or inhibitor or agent)	109604	L12
	L10 and (induced adj proliferation)	127	<u>L11</u>

<u>L10</u>	L2 and (pravastatin or atorvastatin or simvastatin or lovstatin or pindolol or meththiotepin or metoprolol or paldolol)	344	<u>L10</u>
<u>L9</u>	L8 not L7	0	<u>L9</u>
<u>L8</u>	L6 and L2	15	L8
<u>L7</u>	L6 and L5	15	L7
<u>L6</u>	(inhibit or inhibitor or drug or agent) same (induced adj proliferation)	614	<u>L6</u>
<u>L5</u>	L4 and L2	2233	<u>L5</u>
<u>L4</u>	(heart adj valve) same (degeneration or thrombosis or calcification or disease)	2233	<u>L4</u>
<u>L3</u>	(heart adj valve) same (degeneration or throbosis or calcification or disease)	1972	<u>L3</u>
<u>L2</u>	(heart adj valve) or (valvular adj (tissue or endothelial))	8920	L2
<u>L1</u>	Rajamannan-Nalini-M\$.in.	4	<u>L1</u>

# END OF SEARCH HISTORY



# PALM INTRANET

Day: Tuesday
Date: 10/26/2004
Time: 14:19:08

# **Inventor Name Search**

Enter the **first few letters** of the Inventor's Last Name. Additionally, enter the **first few letters** of the Inventor's First name.

Last Name	First Name	
Rajamannan	Nalini Search	

To go back use Back button on your browser toolbar.

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***Beilstein Reactions (File 391)
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***IPA Toxicology (File 153)
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***Toxfile (File 156)
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***Textile Technology Digest (File 119)
     >>> Enter BEGIN HOMEBASE for Dialog Announcements <<<
            of new databases, price changes, etc.
KWIC is set to 50.
HILIGHT set on as ' '
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File
       1:ERIC 1966-2004/Jul 21
       (c) format only 2004 The Dialog Corporation
      Set Items Description
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Cost is in DialUnits
B 155, 5, 73
       26oct04 14:35:04 User259876 Session D684.1
                  0.208 DialUnits File1
            $0.73
     $0.73 Estimated cost File1
     $0.05 INTERNET
     $0.78 Estimated cost this search
     $0.78 Estimated total session cost
                                            0.208 DialUnits
SYSTEM:OS - DIALOG OneSearch
 File 155:MEDLINE(R) 1951-2004/Oct W4
         (c) format only 2004 The Dialog Corp.
 File
         5:Biosis Previews(R) 1969-2004/Oct W3
         (c) 2004 BIOSIS
 File 73:EMBASE 1974-2004/Oct W3
         (c) 2004 Elsevier Science B.V.
     Set Items Description
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S (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR CALCIFICATION)
          1845387 HEART
                  VALVE
          204442
          193224 DEGENERATION
          224910
                  THROMBOSIS
           63559
                  CALCIFICATION
      S1
             269
                  (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR
                  CALCIFICATION)
S S1 (S) (INHIBITOR OR DRUG)
             269 S1
          964040
                  INHIBITOR
         8317261 DRUG
                  S1 (S) (INHIBITOR OR DRUG)
              13
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RD
 ...completed examining records
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T S3/3, K/ALL
  3/3, K/1
              (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
12819432
           PMID: 7586894
 Novel delivery of antiarrhythmic agents.
  Labhasetwar V; Levy R J
  University of Michigan Medical Center, Division of Pediatric Cardiology,
Ann Arbor, USA.
  Clinical pharmacokinetics (NEW ZEALAND)
                                            Jul 1995,
                                                     29 (1) p1-5,
ISSN 0312-5963
               Journal Code: 7606849
  Contract/Grant No.: HL41663; HL; NHLBI
  Document type: Journal Article; Review; Review, Tutorial
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  ... compounds, which are otherwise more potent and less toxic than
available agents. The regional nature of the several cardiac diseases, such
as ischaemia, restenosis or heart valve
                                           calcification , may require a
high concentration of drug at the location of the disease, which by
conventional routes may not be attainable due to systemic toxicity of the
  drug . Localised cardiac delivery of antiarrhythmic agents, based on drug
-polymer implants, may have several advantages, including enhanced drug
 effects and reduced systemic drug toxicity. Computer-assisted automated
feedback systems may further enhance the usefulness of this therapy in the
clinical setting. Before clinical application of this method of drug
delivery further study will be required, but it is hypothesised that
pharmacokinetic variability for drugs delivered in this manner will be
reduced and therefore efficacy...
  3/3, K/2
              (Item 2 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
07972580
          PMID: 3144594
Controlled release of diphosphonates from synthetic polymers to inhibit
calcification.
 Golomb G
 Department
              of Pharmacy, School of Pharmacy,
                                                   Hebrew University of
Jerusalem, Israel.
 Journal of biomaterials applications (UNITED STATES)
                                                         Oct 1987, 2 (2)
p266-89, ISSN 0885-3282 Journal Code: 8813912
```

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... bioprosthetic heart valves fabricated from glutaraldehyde pretreated porcine aortic valves or bovine pericardium. Formulation and evaluation of controlled-release drug delivery system to inhibit bioprosthetic heart valve calcification is reviewed.

3/3,K/3 (Item 3 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

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07567869 PMID: 3116348

Prevention of leaflet calcification of bioprosthetic heart valves with diphosphonate injection therapy. Experimental studies of optimal dosages and therapeutic durations.

Levy R J; Schoen F J; Lund S A; Smith M S

Division of Pediatric Cardiology, C.S. Mott Children's Hospital, University of Michigan Medical Center, Ann Arbor 48109.

Journal of thoracic and cardiovascular surgery (UNITED STATES) Oct 1987

94 (4) p551-7, ISSN 0022-5223 Journal Code: 0376343

Contract/Grant No.: HL32261; HL; NHLBI; HL32346; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... received daily subcutaneous injections of the drug (1, 5, 10, 15, or 25 mg/kg/24 hr) for 21 days with maximal inhibition of bioprosthetic heart valve calcification at a dosage of 15 mg/kg/24 hr (calcium level of diphosphonate-treated bioprostheses 3.5 +/- 0.5 micrograms/ml; calcium level of control...

... mg), but with irreversibly diminished bone and somatic growth. A dosage optimum was observed at 10 mg/kg/24 hr with significant inhibition of bioprosthetic heart valve calcification (at 21 days, the calcium level was 16.4 +/- 3.6 micrograms/mg) and an absence of adverse effects on epiphyseal development and overall growth...

... more calcification after 21 days than did bioprostheses from animals treated for 2 or 3 weeks. Bioprostheses explanted after 110 days from animals receiving the drug (15 mg/kg/24 hr) for the first 3 weeks had calcification equivalent to that of untreated control rats. Diphosphonate (15 mg/kg/24 hr...

3/3,K/4 (Item 4 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

07483079 PMID: 3110404

Controlled-release drug delivery of diphosphonates to inhibit bioprosthetic heart valve calcification: release rate modulation with silicone matrices via drug solubility and membrane coating.

Golomb G; Dixon M; Smith M S; Schoen F J; Levy R J

Journal of pharmaceutical sciences (UNITED STATES) Apr 1987, 76 (4) p271-6, ISSN 0022-3549 Journal Code: 2985195R

Contract/Grant No.: HL32261; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

Controlled-release drug delivery of diphosphonates to inhibit bioprosthetic heart valve calcification: release rate modulation with silicone matrices via drug solubility and membrane coating.

3/3,K/5 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0014260707 BIOSIS NO.: 200300219426

Mechanical prosthetic heart valve thrombosis: Fibrinolysis or surgery? A single center study about 253 patients.

AUTHOR: Roudaut R (Reprint); Lafitte S (Reprint); Roudaut M F (Reprint); Jais C (Reprint); Coste P (Reprint); Roques X (Reprint); Deville C (Reprint); Baudet E (Reprint)

AUTHOR ADDRESS: Hopital Cardiologique, Pessac, France\*\*France
JOURNAL: European Heart Journal 23 (Abstract Supplement): p439

August-September 2002 2002

MEDIUM: print

CONFERENCE/MEETING: Congress of the European Society of Cardiology Berlin,

Germany August 31-September 04, 2002; 20020831

SPONSOR: European Society of Cardiology

ISSN: 0195-668X \_(ISSN print)

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Citation LANGUAGE: English

DESCRIPTORS:

...DISEASES: heart disease, drug therapy, surgery

3/3,K/6 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0008253765 BIOSIS NO.: 199293096656

SCANNING ELECTRON MICROSCOPY STUDIES OF THE PREVENTION OF BIOPROSTHETIC HEART VALVE CALCIFICATION WITH CONTROLLED RELEASE POLYMERIC MATRICES

AUTHOR: PATHAK Y V (Reprint); BOYD J; JOHNSTON T P; LEVY J T; GOLOMB G; SCHOEN F J; LEVY R J

AUTHOR ADDRESS: R-5014 KRESGE II, UNIV MICH, ANN ARBOR, MICH 48109-0576, USA\*\*USA

JOURNAL: Cells and Materials 1 (1): p65-72 1991

ISSN: 1051-6794

DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: ENGLISH

...ABSTRACT: consisting of either ethanehydroxydiphosphonate, FeCl3 or Al(NO3)3 coimplanted with bioprosthetic tissue prevent experimental bioprosthetic calcification. SEM has also been used to study the drug particle distribution in the controlled release matrices. Furthermore, matrix drug release in vitro and in vivo has also been characterized and quantified using SEM techniques.

3/3,K/7 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.

0006962522 BIOSIS NO.: 199039015911

CONTROLLED RELEASE IMPLANTS FOR CARDIOVASCULAR DISEASE

BOOK TITLE: ANDERSON, J. M., S. W. KIM AND K. KNUTSON (ED.). ADVANCES IN

DRUG DELIVERY SYSTEMS, VOL. 4; INTERNATIONAL SYMPOSIUM ON RECENT ADVANCES
IN DRUG DELIVERY SYSTEMS, SALT LAKE CITY, UTAH, USA, FEBRUARY 21-24,

```
1989. X+359P. ELSEVIER SCIENCE PUBLISHERS B.V.: AMSTERDAM, NETHERLANDS;
  NEW YORK, NEW YORK, USA. ILLUS
 AUTHOR: LEVY R J (Reprint); JOHNSTON T P; SINTOV A; GOLOMB G
 AUTHOR ADDRESS: DEP PEDIATR, UNIV MICH, R-5014 KRESGE II, 0576, ANN ARBOR,
   MICH 48109-0576, USA**USA
 SERIES TITLE: Advances in Drug Delivery Systems p245-254 1990
 ISBN: 0-444-88225-1
 DOCUMENT TYPE: Book; Meeting
 RECORD TYPE: Citation
 LANGUAGE: ENGLISH
DESCRIPTORS: DOG RAT SHEEP VENTRICULAR TACHYCARDIA PROSTHETIC HEART VALVE
 CALCIFICATION ETHANEHYDROXYDIPHOSPHONATE CARDIOVASCULAR- DRUG LIDOCAINE
HYDROCHLORIDE ANTIARRHYTHMIC- DRUG
 Set
         Items
                Description
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          269
             FICATION)
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           13
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S3
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S S1 (S) (INDUCED W) PROLIFERATION)
>>>Unmatched parentheses
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                  INDUCED
          522377
                  PROLIFERATION
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               0 S1 (S) (INDUCED (W) PROLIFERATION)
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          964040 INHIBITOR
         8317261 DRUG
          436570 ANTAGONIST
          522377 PROLIFERATION
         1845387 HEART
          204442 VALVE
           50434 HEART (W) VALVE
      S5
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              12
                  (HEART (W) VALVE))
?
...completed examining records
      S6
               9 RD (unique items)
T S6/3, K/ALL
  6/3, K/1
              (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
16932921
           PMID: 15212624
 Sarpogrelate: cardiovascular and renal clinical potential.
 Doggrell Sheila A
            Biomedical Communications, 47
  Doggrell
                                             Caronia Crescent, Lynfield,
Auckland, New Zealand. s.doggrell@xtra.co.nz
 Expert opinion on investigational drugs (England)
                                                         Jul 2004,
p865-74, ISSN 1744-7658
                           Journal Code: 9434197
 Document type: Journal Article
 Languages: ENGLISH
 Main Citation Owner: NLM
 Record type: In Process
 Sarpogrelate is a selective 5-hydroxytryptamine receptor subtype 2A
```

(5-HT2A) antagonist. It is metabolised to racemic M-1 and both enantiomers of M-1 are also antagonists of 5-HT2A receptors. Sarpogrelate inhibits responses to 5-HT mediated by 5-HT2A receptors such as platelet aggregation, vasoconstriction and vascular smooth muscle proliferation. There is no information available on the pharmacokinetics of sarpogrelate. Sarpogrelate is efficacious in animal models of thrombosis, coronary artery spasm, atherosclerosis, restenosis, peripheral vascular...

... disease, myocardial infarction, diabetes and kidney disease. Small clinical trials indicate that sarpogrelate may be beneficial in the treatment of coronary artery disease, angina, restenosis, heart valve prostheses surgery, diabetes mellitus, Raynaud's phenomenon, systemic sclerosis and Buerger's disease. Larger, randomised, double-blind, placebo-controlled clinical trials of sarpogrelate in intermittent...

6/3,K/2 (Item 2 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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14439580 PMID: 10433819

Diminished matrix metalloproteinase 2 (MMP-2) in ectomesenchyme-derived tissues of the Patch mutant mouse: regulation of MMP-2 by PDGF and effects on mesenchymal cell migration.

Robbins J R; McGuire P G; Wehrle-Haller B; Rogers S L

Department of Cell Biology and Physiology, University of New Mexico School of Medicine, 149 Basic Medical Sciences Building, Albuquerque, New Mexico, 87131, USA.

Developmental biology (UNITED STATES) Aug 15 1999, 212 (2) p255-63, ISSN 0012-1606 Journal Code: 0372762

Contract/Grant No.: T32HL07736; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... significantly less. In addition, the migratory ability of branchial arch cells from normal explants could be reduced in a similar manner using a specific MMP inhibitor . Although it is still unclear whether the MMP-2 reduction is a direct result of the absence of response of Ph/Ph cells to PDGF...

6/3,K/3 (Item 3 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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11472750 PMID: 11581595

Operation for anorexigen-associated valvular heart disease.

Caccitolo J A; Connolly H M; Rubenson D S; Orszulak T A; Schaff H V Division of Cardiovascular Surgery and Cardiovascular Diseases, Mayo Clinic, Rochester, MN 55905, USA.

Journal of thoracic and cardiovascular surgery (United States) Oct 2001 122 (4) p656-64, ISSN 0022-5223 Journal Code: 0376343

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... valve replacement, 5 with concomitant aortic valve replacement. Excised valves demonstrated a glistening white appearance with plaque-like encasement of leaflets and chordae. Focal surface proliferation and fibrosis with a "stuck-on" appearance was consistently found. CONCLUSIONS: Anorexigen use may lead to severe multivalvular regurgitation with characteristic echocardiographic and pathologic findings. Recognition of drug -induced valvulopathy is important because of widespread use of these

medications and the uncertain natural history of the disease. Early surgical experience suggests that valve...

6/3,K/4 (Item 4 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

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07035736 PMID: 4090558

[Rational hematologic diagnosis with reference to modern laboratory procedures]

Rationale hamatologische Diagnostik unter Berucksichtigung moderner Laboratoriumsverfahren.

Stobbe H

Zeitschrift fur die gesamte innere Medizin und ihre Grenzgebiete (GERMANY, EAST) Nov 15 1985, 40 (22) p658-60, ISSN 0044-2542 Journal Code: 21730470R

Document type: Journal Article ; English Abstract

Languages: GERMAN

Main Citation Owner: NLM Record type: Completed

The spectre of methods for the diagnostics and differentiation of haemolytic anaemias, particularly for the establishment of congenital, autoimmune haemolytic, **drug** -conditioned and other anaemias is treated. The clear delimitation of an iron deficiency from a disturbance of the iron distribution is advantageously to be achieved...

... haematopoietic stem cells are particularly evident in the aplastic syndrome of the bone marrow and further haematological diseases concerning the establishment of the intensiveness of **proliferation**. The classification of the acute leukemias demands conventional as well as cytochemical staining methods; recently, it is essentially improved using monoclonal antibodies. In leukemias cytogenetic...

#### 6/3,K/5 (Item 5 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

06804443 PMID: 3922293

Influence of the developmental state of valvular lesions on the antimicrobial activity of cefotaxime in experimental enterococcal infections.

Sullam P M; Drake T A; Tauber M G; Hackbarth C J; Sande M A Antimicrobial agents and chemotherapy (UNITED STATES) Mar 1985, 27 (3) p320-3, ISSN 0066-4804 Journal Code: 0315061

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Descriptors: Cefotaxime--therapeutic use--TU; \*Endocarditis, Bacterial --drug therapy--DT; \*Heart Valve Diseases-- **drug** therapy--DT; \*Streptococcal Infections--drug therapy--DT

#### 6/3,K/6 (Item 1 from file: 73)

DIALOG(R) File 73:EMBASE

(c) 2004 Elsevier Science B.V. All rts. reserv.

12166070 EMBASE No: 2003270282

NFATc1 mediates vascular endothelial growth factor-induced proliferation of human pulmonary valve endothelial cells

Johnson E.N.; Lee Y.M.; Sander T.L.; Rabkin E.; Schoen F.J.; Kaushal S.;

Bischoff J.

J. Bischoff, Dept. of Surgery, Children's Hospital, 300 Longwood Ave., Boston, MA 02115 United States

AUTHOR EMAIL: joyce.bischoff@tch.harvard.edu

Journal of Biological Chemistry ( J. BIOL. CHEM. ) (United States) 17 JAN 2003, 278/3 (1686-1692)

CODEN: JBCHA ISSN: 0021-9258 DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 36

...endothelial growth factor (VEGF) signaling through VEGF receptor 2. VEGF-induced NFATc1 nuclear translocation was inhibited by either cyclosporin A or a calcineurin-specific peptide inhibitor; these findings suggest that VEGF stimulates NFATc1 nuclear import in human pulmonary valve endothelial cells by a calcineurin-dependent mechanism. Importantly, both cyclosporin A and the calcineurin-specific peptide inhibitor reduced VEGF-induced human pulmonary valve endothelial cell proliferation, indicating a functional role for NFATc1 in endothelial growth. In contrast, VEGF-induced proliferation of human dermal microvascular and human umbilical vein endothelial cells was not sensitive to cyclosporin A. Finally, NFATc1 was detected in the endothelium of human...

...valve leaflets by immunohistochemistry. These results suggest VEGF-induced NFATcl activation may be an important mechanism in cardiac valve maintenance and function by enhancing endothelial **proliferation** 

6/3,K/7 (Item 2 from file: 73)
DIALOG(R)File 73:EMBASE

(c) 2004 Elsevier Science B.V. All rts. reserv.

11742403 EMBASE No: 2002318036

Future potential indications for an oral thrombin inhibitor ZUKUNFTIQES INDIKATIONSPOTENZIAL EINES ORALEN THROMBININHIBITORS Haas S.

Dr. S. Haas, Inst. Exp. Onkol./Therapieforschung, Technische Universitat Munchen, Ismaninger Str. 22, 81675 Munchen Germany

AUTHOR EMAIL: sylvia.haas@lrz.tum.de

Hamostaseologie ( HAMOSTASEOLOGIE ) (Germany) 2002, 22/3 (118-125)

CODEN: HAEMD ISSN: 0720-9355 DOCUMENT TYPE: Journal; Review

LANGUAGE: GERMAN SUMMARY LANGUAGE: ENGLISH; GERMAN

NUMBER OF REFERENCES: 35

...with food ingredients and drugs. The search for new antithrombotics with an improved safety/efficacy profile led to the development of the direct oral thrombin- inhibitor ximelagatran. It can be administered without routine monitoring of coagulation parameters and does not possess any of the previously mentioned limitations. The results from clinical...

...as an alternative anticoagulant in heparin induced thrombocytopenia and for prevention of thromboembolic complications in oncology. Because of the mitogenic effects of thrombin on the **proliferation** of tumour cells, additional experimental studies aiming at a potential inhibition of thrombin-triggered oncogenesis is of uttermost interest.

6/3,K/8 (Item 3 from file: 73)

DIALOG(R) File 73:EMBASE

(c) 2004 Elsevier Science B.V. All rts. reserv.

11419458 EMBASE No: 2001432739

Cell proliferation in carcinoid valve disease: A mechanism for serotonin effects

Rajamannan N.M.; Caplice N.; Anthikad F.; Sebo T.J.; Orszulak T.A.; Edwards W.D.; Tajik J.; Schwartz R.S. Dr. N.M. Rajamannan, Northwestern University Medical Sch., Division of Cardiology, 250 East Superior Street, Chicago, IL 60611 United States Journal of Heart Valve Disease ( J. HEART VALVE DIS. ) (United Kingdom) 2001, 10/6 (827-831) CODEN: JHVDE ISSN: 0966-8519 DOCUMENT TYPE: Journal ; Article LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH NUMBER OF REFERENCES: 17

...proliferative effects of serotonin (10SUP-8to 10SUP-6M) on cultured porcine aortic valve cells via a [SUP3H] thymidine assay were determined in vitro. Serotonin receptor antagonist studies in culture were also performed using methiotepin, a 5HTSUB1b antagonist , and ketanserin, a 5HTSUB2 receptor antagonist , to determine the mechanism of serotonin action. The ex-vivo proliferation level in human carcinoid (n = 26) and normal valves (n = 10) was compared using proliferating cell nuclear antigen (PCNA) staining, a marker for proliferation . Identification and localization of specific 5HT receptor was assessed by immunostaining for serotonin receptors in the valves. Results: Serotonin increased valvular proliferation in vitro in a dose-dependent manner (10-fold increase) (p <0.001), and this mitogenic effect was inhibited by methiotepin but not ketanserin. In human carcinoid heart valves the level of proliferation was 35-fold higher than in normal human valves (p <0.001). 5HTSUB1b receptors were found only in the carcinoid valves, and not in the normal valves. Conclusion: Serotonin is a powerful mitogen for valvular subendocardial cells. The mitogenic effect is at least partly mediated via 5HTSUB1b receptors. Subendothelial cell proliferation is significantly elevated in human carcinoid valves in vivo. The data suggest a mechanism whereby serotonin may contribute to valvular proliferation in carcinoid heart disease.

```
6/3, K/9
               (Item 4 from file: 73)
DIALOG(R) File 73:EMBASE
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EMBASE No: 1979216176
```

Some recent advances in cardiac pathology

Billingham M.E.

Dept. Pathol., Stanford Univ. Sch. Med., Stanford, Calif. United States Human Pathology ( HUM. PATHOL. ) (United States) 1979, 10/4 (367-386) CODEN: HPCQA

DOCUMENT TYPE: Journal

LANGUAGE: ENGLISH

This article has reviewed some recent advances in human cardiac pathology. The areas selected were cardiac transplant pathology, the lesions of drug cardiotoxicity, and the morphology of tissue heart valve replacements. The proliferation of work in the first two areas is largely the result of the successful introduction of a new procedure, endomyocardial biopsy, which has made it...

```
Set
        Items
                Description
S1
                (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR CALCI-
          269
             FICATION)
S2
           13
                S1 (S) (INHIBITOR OR DRUG)
S3
                RD (unique items)
            7
S4
            Ω
                S1 (S) (INDUCED (W) PROLIFERATION)
S5
           12
                (INHIBITOR OR DRUG OR ANTAGONIST) (S) (PROLIFERATION AND (-
            HEART (W) VALVE))
S6
            9
                RD (unique items)
```

```
S (SCREENING OR IDENTIFYING) (S) (INHIBITORS OR DRUGS OR ANTAGONISTS)
           546440 SCREENING
           139087
                   IDENTIFYING
           834708
                  INHIBITORS
           954099 DRUGS
           563388
                  ANTAGONISTS
      S7
           28060
                   (SCREENING OR IDENTIFYING) (S) (INHIBITORS OR DRUGS OR
                   ANTAGONISTS)
S S1 AND S7
              269 S1
            28060 S7
      S8
               1 S1 AND S7
T S8/3, K/ALL
  8/3,K/1
               (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
(c) 2004 BIOSIS. All rts. reserv.
0014679298
             BIOSIS NO.: 200400060055
 Bioprosthetic heart valves
AUTHOR: Rajamannan Nalini M (Reprint)
AUTHOR ADDRESS: Rochester, MN, USA**USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office
Patents 1277 (2): Dec. 9, 2003 2003
MEDIUM: e-file
PATENT NUMBER: US 6660260 PATENT DATE GRANTED: December 09, 2003 20031209
PATENT CLASSIFICATION: 424-9321 PATENT ASSIGNEE: Mayo Foundation for
Medical Education and Research PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English
... ABSTRACT: heart valve cells and heart valve cusps as well as methods for
  making heart valves. The invention also provides methods and materials
  for (1) slowing heart
                         valve
                                 degeneration , thrombosis, and
  calcification, (2) treating carcinoid heart disease, (3) identifying
 inhibitors of heart
                      valve
                                degeneration , thrombosis, and
  calcification, and (4) determining the safety of drugs
DESCRIPTORS:
  ...DISEASES: heart
                       valve
                                degeneration --
Set
        Items
                Description
S1
          269
                (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR CALCI-
             FICATION)
S2
           13
                S1 (S) (INHIBITOR OR DRUG)
                RD (unique items)
s_3
S4
            0
                S1 (S) (INDUCED (W) PROLIFERATION)
$5
           12
                (INHIBITOR OR DRUG OR ANTAGONIST) (S) (PROLIFERATION AND (-
             HEART (W) VALVE))
S<sub>6</sub>
            9
                RD (unique items)
S7
                (SCREENING OR IDENTIFYING) (S) (INHIBITORS OR DRUGS OR ANT-
        28060
             AGONISTS)
S8
                S1 AND S7
?
S (HEART (W) VALVE) (S) (PROLIFERATION)
         1845387 HEART
          204442 VALVE
          522377 PROLIFERATION
      59
              56 (HEART (W) VALVE) (S) (PROLIFERATION)
S S7 AND S9
```

```
28060 S7
             56 S9
0 S7 AND S9
     S10
?
S S9 AND (INHIBITOR? OR ANTAGONIST? OR DRUG?)
Processing
              56 S9
         1935976
                 INHIBITOR?
          897063 ANTAGONIST?
         8640436 DRUG?
              14 S9 AND (INHIBITOR? OR ANTAGONIST? OR DRUG?)
     S11
?
RD
...completed examining records
               8 RD (unique items)
T S12/3, K/ALL
  12/3, K/1
               (Item 1 from file: 155)
DIALOG(R) File 155: MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
16932921
           PMID: 15212624
 Sarpogrelate: cardiovascular and renal clinical potential.
  Doggrell Sheila A
             Biomedical Communications, 47 Caronia Crescent, Lynfield,
Auckland, New Zealand. s.doggrell@xtra.co.nz
  Expert opinion on investigational drugs (England)
                                                        Jul 2004, 13 (7)
 p865-74, ISSN 1744-7658
                           Journal Code: 9434197
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: In Process
  Sarpogrelate is a selective 5-hydroxytryptamine receptor subtype 2A
(5-HT2A)
          antagonist . It is metabolised to racemic M-1 and both
enantiomers of M-1 are also antagonists of 5-HT2A receptors. Sarpogrelate
inhibits responses to 5-HT mediated by 5-HT2A receptors such as platelet
aggregation, vasoconstriction and vascular smooth muscle proliferation...
... disease, myocardial infarction, diabetes and kidney disease. Small
clinical trials indicate that sarpogrelate may be beneficial in the
treatment of coronary artery disease, angina, restenosis, heart
prostheses surgery, diabetes mellitus, Raynaud's phenomenon, systemic
                            disease. Larger, randomised, double-blind,
sclerosis
          and
                 Buerger's
placebo-controlled clinical trials of sarpogrelate in intermittent...
  12/3, K/2
               (Item 2 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.
14439580
          PMID: 10433819
Diminished matrix metalloproteinase 2 (MMP-2) in ectomesenchyme-derived
 tissues of the Patch mutant mouse: regulation of MMP-2 by PDGF and effects
on mesenchymal cell migration.
 Robbins J R; McGuire P G; Wehrle-Haller B; Rogers S L
 Department of Cell Biology and Physiology, University of New Mexico
School of Medicine, 149 Basic Medical Sciences Building, Albuquerque, New
Mexico, 87131, USA.
 Developmental biology (UNITED STATES)
                                         Aug 15 1999, 212 (2) p255-63,
ISSN 0012-1606
                Journal Code: 0372762
 Contract/Grant No.: T32HL07736; HL; NHLBI
 Document type: Journal Article
 Languages: ENGLISH
```

Main Citation Owner: NLM Record type: Completed

... receptor. Homozygous (Ph/Ph) embryos exhibit multiple connective tissue defects including cleft face (involving the first branchial arch and frontonasal processes), incomplete heart septation, and heart valve abnormalities before they die in utero. Analyses of the cell biology underlying the defects in Ph/Ph embryos have revealed a deficit in a matrix ...

...significantly less. In addition, the migratory ability of branchial arch cells from normal explants could be reduced in a similar manner using a specific MMP **inhibitor**. Although it is still unclear whether the MMP-2 reduction is a direct result of the absence of response of Ph/Ph cells to PDGF...

; Animals; Branchial Region--cytology--CY; Branchial Region--embryology
--EM; Branchial Region--enzymology--EN; Face--embryology--EM; Gelatinase A
; Gelatinases-- antagonists and inhibitors --AI; Heart--embryology--EM;
Mesoderm--enzymology--EN; Metalloendopeptidases-- antagonists and
inhibitors --AI; Mice; Mice, Mutant Strains; Morphogenesis; Myocardium
--enzymology--EN; Skull--embryology--EM; Tissue Culture

12/3,K/3 (Item 3 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

13118099 PMID: 8784007

Impact of glutaraldehyde on calcification of pericardial bioprosthetic heart valve material.

Grabenwoger M; Sider J; Fitzal F; Zelenka C; Windberger U; Grimm M; Moritz A; Bock P; Wolner E

Department of Cardio-Thoracic Surgery, University of Vienna, Austria. Annals of thoracic surgery (UNITED STATES) Sep 1996, 62 (3) p772-7, ISSN 0003-4975 Journal Code: 15030100R

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... by dye-mediated photooxidation showed no calcification after 63 days of subcutaneous implantation (1.0 +/- 0.4 mg Ca2+/g dry weight). Regular endothelial cell **proliferation** was observed on photooxidized and L-glutamic acid-treated tissue, whereas conventionally treated tissue caused endothelial cell death. CONCLUSIONS: This study underlines the detrimental role of glutaraldehyde in the calcification process of bioprosthetic **heart valve** materials and emphasizes alternative preservation methods that reduce or avoid the use of glutaraldehyde.

Descriptors: Bioprosthesis; \*Calcinosis--pathology--PA; \*Glutaral --pharmacology--PD; \*Heart Valve Prosthesis; \*Pericardium-- **drug** effects --DE

12/3,K/4 (Item 4 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

11208023 PMID: 11245774

The genetics and physiology of polycystic kidney disease.

Calvet J P; Grantham J J

Department of Biochemistry, Kidney Institute, University of Kansas Medical Center, Kansas City, KS, USA. jcalvet@kumc.edu

Seminars in nephrology (United States) Mar 2001, 21 (2) p107-23,

ISSN 0270-9295 Journal Code: 8110298

Document type: Journal Article; Review; Review Literature

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... 1 transcription factor. In addition, polycystin-2 may function in mediating calcium flux. The pathogenesis of cyst formation is currently thought to involve increased cell **proliferation**, fluid accumulation, and basement membrane remodeling. It now appears that cyclic adenosine monophosphate (cAMP) metabolism is a central component of cyst formation, stimulating apical chloride...

... cyst fluid. Recent evidence has shown that ADPKD cells also have an altered responsiveness to cyclic AMP. In contrast to normal kidney cells whose cell **proliferation** is inhibited by cyclic AMP, ADPKD cells are stimulated to proliferate. Thus, it is likely that an alteration in polycystin function transforms the normal cellular phenotype to one that responds to elevated cyclic AMP by an increased rate of cell **proliferation** and that the enlarging cyst expands by an increased rate of cyclic AMP-driven fluid secretion. Cyclic AMP and growth factors, including epidermal growth factor...

... effects to accelerate the enlargement of ADPKD cysts, and thereby to contribute to the progression of the disease. This knowledge should facilitate the discovery of **inhibitors** of signal transduction cascades that can be used in the treatment of ADPKD. Copyright 2001 by W.B. Saunders Company

12/3,K/5 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2004 The Dialog Corp. All rts. reserv.

09132515 PMID: 1728078

Glutaraldehyde affects biocompatibility of bioprosthetic heart valves.

Grimm M; Eybl E; Grabenwoger M; Spreitzer H; Jager W; Grimm G; Bock P; Muller M M; Wolner E

Second Department of Surgery, University of Vienna, Austria.

Surgery (UNITED STATES) Jan 1992, 111 (1) p74-8, ISSN 0039-6060

Journal Code: 0417347

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

... washing solutions was found by high performance liquid chromatography (up to 1.8 ppm of glutaraldehyde per gram of dry tissue). In vitro endothelial cell **proliferation** rate was impaired dose-dependently in the presence of increasing glutaraldehyde concentrations of the cultivation medium (r = 0.9; p less than 0.05). Cultivation...

; Animals; Cattle; Cell Division-- **drug** effects--DE; Cells, Cultured; Endothelium, Vascular--cytology--CY; Glutaral--analysis--AN; Materials Testing

12/3,K/6 (Item 1 from file: 73)
DIALOG(R)File 73:EMBASE
(c) 2004 Elsevier Science B.V. All rts. reserv.

12788379 EMBASE No: 2004382559

Heart valve development: Endothelial cell signaling and differentiation

Armstrong E.J.; Bischoff J.

Dr. J. Bischoff, Vascular Biology Program, Children's Hospital Boston, 300 Longwood Ave, Boston, MA 02115 United States

AUTHOR EMAIL: joyce.bischoff@childrens.harvard.edu

Circulation Research ( CIRC. RES. ) (United States) 03 SEP 2004, 95/5

(459 - 470)

CODEN: CIRUA ISSN: 0009-7330 DOCUMENT TYPE: Journal; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 99

...beta-catenin, BMP/TGF-beta, ErbB, and NF1/Ras. Based on the interactions among and relative timing of these pathways, a signaling network model for **heart valve** development is proposed. DRUG DESCRIPTORS:

...epidermal growth factor receptor 2--endogenous compound--ec; epidermal growth factor receptor 3--endogenous compound--ec; epidermal growth factor receptor 4--endogenous compound--ec; unclassified **drug** 

12/3,K/7 (Item 2 from file: 73)

DIALOG(R)File 73:EMBASE

(c) 2004 Elsevier Science B.V. All rts. reserv.

05146863 EMBASE No: 1992287096

Endothelial cell lining of bioprosthetic heart valve materials

Eybl E.; Grimm M.; Grabenwoger M.; Bock P.; Muller M.M.; Wolner E. Second Department of Surgery, Zentrallabor B 800, University of Vienna, Spitalgasse 23,A-1090 Vienna Austria

Journal of Thoracic and Cardiovascular Surgery ( J. THORAC. CARDIOVASC.

SURG. ) (United States) 1992, 104/3 (763-769)

CODEN: JTCSA ISSN: 0022-5223 DOCUMENT TYPE: Journal; Article

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

...of antithrombogenic potency of the seeded cells on L-glutamic acid-treated valve material was proved by regular release of prostacyclin. We conclude that bioprosthetic **heart** valve materials can be lined with endothelial cells if toxic glutaraldehyde released from the bioprostheses is eliminated.

DRUG DESCRIPTORS:

collagen; fibronectin; glutamic acid; glutaraldehyde-- drug toxicity--to; prostacyclin--endogenous compound--ec

12/3,K/8 (Item 3 from file: 73)

DIALOG(R) File 73: EMBASE

(c) 2004 Elsevier Science B.V. All rts. reserv.

01495127 EMBASE No: 1979216176

Some recent advances in cardiac pathology

Billingham M.E.

Dept. Pathol., Stanford Univ. Sch. Med., Stanford, Calif. United States Human Pathology ( HUM. PATHOL. ) (United States) 1979, 10/4 (367-386)

CODEN: HPCQA

DOCUMENT TYPE: Journal

LANGUAGE: ENGLISH

This article has reviewed some recent advances in human cardiac pathology. The areas selected were cardiac transplant pathology, the lesions of **drug** cardiotoxicity, and the morphology of tissue **heart valve** replacements. The **proliferation** of work in the first two areas is largely the result of the successful introduction of a new procedure, endomyocardial biopsy, which has made it...
DRUG DESCRIPTORS:

\* drug

MEDICAL DESCRIPTORS:

electron microscopy; histology; immunology; heart; adverse **drug** reaction; review

?

```
Set
         Items
                 Description
 S1
           269
                 (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR CALCI-
              FICATION)
 S2
            13
                 S1 (S) (INHIBITOR OR DRUG)
 S3
                 RD (unique items)
 S4
             0
                 S1 (S) (INDUCED (W) PROLIFERATION)
 S5
            12
                 (INHIBITOR OR DRUG OR ANTAGONIST) (S) (PROLIFERATION AND (-
              HEART (W) VALVE))
                 RD (unique items)
S6
S7
         28060
                 (SCREENING OR IDENTIFYING) (S) (INHIBITORS OR DRUGS OR ANT-
              AGONISTS)
S8
                 S1 AND S7
             1
                 (HEART (W) VALVE) (S) (PROLIFERATION)
S9
            56
S10
                 S7 AND S9
             0
S11
            14
                 S9 AND (INHIBITOR? OR ANTAGONIST? OR DRUG?)
S12
             8
                 RD (unique items)
S S7 AND (PRAVASTATIN OR ATORVASTATIN OR SIMVASTATIN OR LOVSTATIN OR PINDOLOL OR MET
            28060 S7
            10542 PRAVASTATIN
             6824
                   ATORVASTATIN
            13403
                  SIMVASTATIN
                7 LOVSTATIN
            16525 PINDOLOL
                0 METHTHIOTEPIN
            25629 METOPROLOL
                0
                  PALDOLOL
     S13
              277
                   S7 AND (PRAVASTATIN OR ATORVASTATIN OR SIMVASTATIN OR
                   LOVSTATIN OR PINDOLOL OR METHTHIOTEPIN OR METOPROLOL OR
                   PALDOLOL)
S S1 AND S13
              269
                  S1
              277
                  $13
                  S1 AND S13
     S14
                0
?
S S13 AND (HEART (W) VALVE)
             277
                  S13
         1845387
                  HEART
          204442
                  VALVE
           50434
                  HEART (W) VALVE
     S15
               0 S13 AND (HEART (W) VALVE)
?
S (INHIBITED OR REDUCED) (S) (PROLIFERATION AND (HEART (W) VALVES))
          912838 INHIBITED
         1711980 REDUCED
          522377
                  PROLIFERATION
         1845387
                  HEART
           41098
                  VALVES
            9808 HEART (W) VALVES
     S16
              18
                  (INHIBITED OR REDUCED) (S) (PROLIFERATION AND (HEART (W)
                  VALVES))
S S13 AND S16
             277
                  S13
              18
                  S16
     S17
                  S13 AND S16
               0
?
RD S18
>>>Set 18 has not yet been created.
RD S16
...completed examining records
               8 RD S16 (unique items)
```

? T S18/3, K/ALL

18/3,K/1 (Item 1 from file: 155) DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

14290451 PMID: 10194805

Bisdiamine inhibits extracellular matrix formation and cell proliferation of atrioventricular mesenchyme from developing chick heart valves.

Choy M; Oltjen S L; Moon A J; Armstrong M T; Armstrong P B

Division of Pediatric Cardiology, University of California Davis Medical Center, Sacramento 95817, USA. mchoy@ucdavis.edu

Teratology (UNITED STATES) Mar 1999, 59 (3) p148-55, ISSN 0040-3709

Journal Code: 0153257

Document type: Journal Article

Languages: ENGLISH Main Citation Owner: NLM Record type: Completed

... on mesenchymal cells grown in aggregate culture isolated from the developing atrioventricular valves of the stage-36 chick embryo. Fibronectin extracellular matrix formation and cell proliferation in the aggregates were assessed in various media. Chick serum stimulated the cells to produce an extracellular matrix and to divide, and the inclusion of bisdiamine inhibited both responses. If we isolated an extracellular matrix from a monolayer of mesenchymal cells and added the sonicated matrix to the medium containing serum and...

... attach to an intact extracellular matrix to begin cell division. Thus, bisdiamine inhibits the normal formation of the we suggest that extracellular matrix, leading to reduced cell proliferation , but it does not affect matrix-cell interaction. The lack of cushion growth in situ may be the cause of AVSD or TA.

18/3, K/2(Item 2 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

14210123 PMID: 9930452

Effect of antibiotic pretreatment on immunogenicity of human heart valves and component cells.

Johnson D L; Sloan C; O'Halloran A; Yacoub M H

Department of Cardiothoracic Surgery, National Heart and Lung Institute, Imperial College at Harefield Hospital, Heart Science Centre, Middlesex, United Kingdom.

Annals of thoracic surgery (UNITED STATES) Dec 1998, 66 (6 Suppl) pS221-4, ISSN 0003-4975 Journal Code: 15030100R

Document type: Journal Article

Languages: ENGLISH Main Citation Owner: NLM Record type: Completed

... low-dose antibiotics, peripheral blood mononuclear cells and human allogenic T cells were cocultured with antibiotic-treated valve discs, cultured valve endothelial cells, and fibroblasts. Proliferation was measured by uptake of thymidine labeled with hydrogen 3. RESULTS: Untreated tissue pieces stimulate peripheral blood mononuclear cells (4,080+/-980 cpm) at day...

... study shows that valve tissue is immunogenic and this immunogenicity is mediated mainly by endothelial cells. However, the immunostimulatory potential of the valve can be reduced by incubating the solution in an antibiotic cocktail.

18/3,K/3 (Item 3 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

13165284 PMID: 8834727

In vitro endothelialization of bioprosthetic heart valves.

Fischlein T; Fasol R

Department of Cardiac Surgery Grosshadern Medical Center, University of Munich, Germany.

Journal of heart valve disease (ENGLAND) Jan 1996, 5 (1) p58-65,

ISSN 0966-8519 Journal Code: 9312096

Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... to conventional bioprosthetic heart valves, glutaraldehyde preserved porcine aortic valve leaflets were precoated with fibronectin-heparin and acidic fibroblast growth factor (aFGF) to enhance cell **proliferation**. Furthermore, different methods of storage and preservation (1.0% benzoic acid, 1.0% sorbic acid, 0.05% and 0.5% dialdehyde starch) were compared to ...

...aFGF protein and endothelial cells improved in vitro and in vivo results significantly. CONCLUSIONS: Our study shows that endothelial cell growth as well as significantly reduced in vivo degeneration and mineralization of valve leaflets may be feasible if bioprosthetic heart valves are processed according to alternative, non-toxic conservation procedures and are precoated with angiogenic growth factor protein.

18/3,K/4 (Item 4 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

13103742 PMID: 8725286

Fibroblast growth factor-2 stimulates embryonic cardiac mesenchymal cell proliferation.

Choy M; Oltjen S L; Otani Y S; Armstrong M T; Armstrong P B
Division of Pediatric Cardiology, University of California Davis Medical
Center, Sacramento 95817, USA.

Developmental dynamics - an official publication of the American Association of Anatomists (UNITED STATES) Jun 1996, 206 (2) p193-200, ISSN 1058-8388 Journal Code: 9201927

Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM

Record type: Completed

The **proliferation** response of stage 36 chick atrioventricular valve mesenchymal cells to fibroblast growth factor-2 (FGF-2) was studied in the tissue-like environment of three...

inclusion of Arg-Gly-Asp-containing peptides, which compete with fibronectin for binding to the cell surface alpha 5 beta 1 integrin receptors, abolished the **proliferation** effects of FGF-2. Inhibition of sulfation of cell surface glycosaminoglycans by treatment with sodium chlorate significantly **reduced** both the formation of the fibronectin matrix and cell **proliferation** in response to FGF-2, suggesting an involvement of the low-affinity sulfated glycosaminoglycan FGF receptor system. Thus, the FGF-stimulated growth of embryonic atrioventricular...

18/3,K/5 (Item 5 from file: 155)
DIALOG(R) File 155:MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

12387819 PMID: 12773386

Defective valvulogenesis in HB-EGF and TACE-null mice is associated with aberrant BMP signaling.

Jackson Leslie F; Qiu Ting Hu; Sunnarborg Susan W; Chang Aileen; Zhang Chunlian; Patterson Cam; Lee David C

Department of Biochemistry & Biophysics, UNC Lineberger Comprehensive Cancer Center, University of North Carolina School of Medicine, Chapel Hill, NC 27599, USA.

EMBO journal (England) Jun 2 2003, 22 (11) p2704-16, ISSN 0261-4189 Journal Code: 8208664

Contract/Grant No.: AG021096; AG; NIA; CA43793; CA; NCI; CA61896; CA; NCI; CA85410; CA; NCI; HL65619; HL; NHLBI

Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... double null HB-EGF(-/-)/BTC(-/-) mice was further reduced, apparently due to accelerated heart failure. HB-EGF(-/-) newborns had enlarged and malformed semilunar and atrioventricular heart valves, and hypoplastic, poorly differentiated lungs. Defective cardiac valvulogenesis was the result of abnormal mesenchymal cell proliferation during remodeling, and was associated with dramatic increases in activated Smad1/5/8. Consistent with the phenotype, HB-EGF transcripts were localized to endocardial cells...

18/3,K/6 (Item 6 from file: 155)

DIALOG(R) File 155:MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

11585869 PMID: 11767194

Cell proliferation in carcinoid valve disease: a mechanism for serotonin effects.

Rajamannan N M; Caplice N; Anthikad F; Sebo T J; Orszulak T A; Edwards W D; Tajik J; Schwartz R S

Division of Cardiovascular Diseases and Internal Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota, USA.

Journal of heart valve disease (England) Nov 2001, 10 (6) p827-31, ISSN 0966-8519 Journal Code: 9312096

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM Record type: Completed

...culture were also performed using methiotepin, a 5HT1b antagonist, and ketanserin, a 5HT2 receptor antagonist, to determine the mechanism of serotonin action. The ex-vivo proliferation level in human carcinoid ( $n = \frac{1}{2}$ and normal valves (n = 10) was compared using proliferating cell antigen staining, (PCNA) a marker for **proliferation** . Identification and localization of specific 5HT receptor was assessed by immunostaining for serotonin receptors in the valves. RESULTS: Serotonin increased valvular proliferation in vitro in a dose-dependent manner (10-fold increase) (p < 0.001), and this mitogenic effect was **inhibited** by methiotepin but not ketanserin. In human carcinoid heart level of proliferation was 35-fold higher than in normal human valves (p <0.001). 5HT1b receptors were found only in the carcinoid valves, and not in the normal valves. CONCLUSION: Serotonin is a powerful mitogen for valvular subendocardial cells. The mitogenic effect is at least partly mediated via 5HT1b receptors. Subendothelial cell proliferation is significantly elevated in human carcinoid valves in vivo. The data suggest a mechanism whereby serotonin may contribute to valvular proliferation in carcinoid heart disease.

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18/3,K/7 (Item 7 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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(c) format only 2004 The Dialog Corp. All rts. reserv.

10289041 PMID: 7986560

Integrity and viability of homograft valves.

Fischlein T; Schutz A; Uhlig A; Frey R; Krupa W; Babic R; Thiery J; Reichart B

Department of Cardiac Surgery, University of Munich, Germany.

European journal of cardio-thoracic surgery - official journal of the European Association for Cardio-thoracic Surgery (GERMANY) 1994, 8 (8) p425-30, ISSN 1010-7940 Journal Code: 8804069

Comment in Eur J Cardiothorac Surg. 1995;9(2) 113; Comment in PMID 7748572

Document type: Journal Article

Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed

... 3 days beforehand. Morphological observations were made using light and electron microscopy and, in order to characterize the endothelial cell viability, a non-radioactive cell **proliferation** assay was used. The PGI2 secretion of the remaining endothelium was defined as the 6-keto-prostaglandin F1 alpha metabolite by an enzyme immunoassay. Observations...

... of HBD homografts was maintained was confirmed by proven PGI2 secretion (6150 +/- 1200 pg/3 ml M199 after cryopreservation), whereas specimens from NHBD showed a reduced (1950 +/- 730 pg/3 ml M199) and, after cryopreservation, almost no release (P < 0.0001).(ABSTRACT TRUNCATED AT 250 WORDS)

#### 18/3,K/8 (Item 8 from file: 155)

DIALOG(R) File 155: MEDLINE(R)

(c) format only 2004 The Dialog Corp. All rts. reserv.

07260406 PMID: 3490326

Porcine heart valves produce a protein that induces cell-mediated connective tissue degradation: II. Biochemical properties of the partially purified protein.

Decker R S; Henney A M; Dingle J T

Circulation research (UNITED STATES) Sep 1986, 59 (3) p329-41,

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

...release of glycosaminoglycans from cultured cartilage and mitral valve and provoked porcine valves to degrade their own collagen extracellular matrix. The release of hydroxyproline was **inhibited** by corticosteroids, whereas proteoglycan breakdown was not. Partially pure preparations of CCF and synovial catabolin stimulated murine thymocyte **proliferation**; moreover, that activity was almost totally abolished by an antibody raised against pure porcine interleukin-1. These observations suggest that CCF may represent a catabolic...

```
Set Items Description
```

- S1 269 (HEART (W) VALVE) (W) (DEGENERATION OR THROMBOSIS OR CALCI-FICATION)
- S2 13 S1 (S) (INHIBITOR OR DRUG)
- S3 7 RD (unique items)

```
S1 (S) (INDUCED (W) PROLIFERATION)
S5
                 (INHIBITOR OR DRUG OR ANTAGONIST) (S) (PROLIFERATION AND (-
           12
             HEART (W) VALVE))
S6
                RD (unique items)
S7
        28060
                 (SCREENING OR IDENTIFYING) (S) (INHIBITORS OR DRUGS OR ANT-
             AGONISTS)
S8
            1
                S1 AND S7
S9
           56
                (HEART (W) VALVE) (S) (PROLIFERATION)
S10
            Ω
                S7 AND S9
S11
                S9 AND (INHIBITOR? OR ANTAGONIST? OR DRUG?)
           14
S12
            8
                RD (unique items)
S13
                S7 AND (PRAVASTATIN OR ATORVASTATIN OR SIMVASTATIN OR LOVS-
          277
             TATIN OR PINDOLOL OR METHTHIOTEPIN OR METOPROLOL OR PALDOLOL)
S14
                S1 AND S13
S15
                S13 AND (HEART (W) VALVE)
            0
S16
                (INHIBITED OR REDUCED) (S) (PROLIFERATION AND (HEART (W) V-
           18
             ALVES))
S17
            Ω
                S13 AND S16
S18
            8
                RD S16 (unique items)
?
COST
       26oct04 15:06:34 User259876 Session D684.2
            $6.31
                     1.971 DialUnits File155
               $4.62 22 Type(s) in Format 3
            $4.62 22 Types
    $10.93 Estimated cost File155
            $7.54
                     1.347 DialUnits File5
               $7.00 4 Type(s) in Format 3
            $7.00 4 Types
   $14.54 Estimated cost File5
           $27.62
                    2.819 DialUnits File73
              $18.90 7 Type(s) in Format 3
           $18.90 7 Types
   $46.52 Estimated cost File73
           OneSearch, 3 files, 6.137 DialUnits FileOS
    $8.00
           INTERNET
   $79.99
           Estimated cost this search
   $80.77 Estimated total session cost
                                           6.345 DialUnits
```

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